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SLEEP DEPRIVATION AND SUSTAINED OPERATIONS: EFFECTS ON INDICES --ETC(U)
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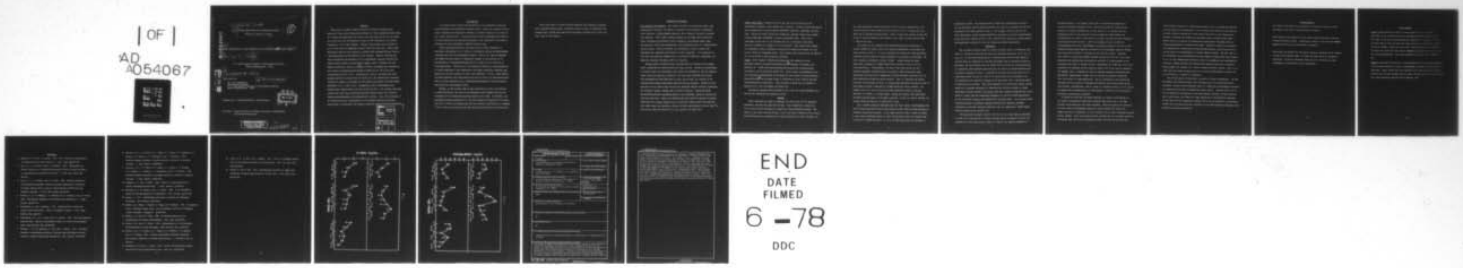

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Sleep Deprivation and Sustained Operations: Effects on Indices of Stress

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Abstract

Two groups of highly trained and motivated military personnel were deprived of sleep while sustaining performance of their assigned military tasks in a laboratory simulation; one team (I) was sleep deprived for 48 hours while the second team (II) was deprived of sleep for two consecutive 39 h periods separated by a 33 h rest interval. Six-hour urine samples were collected on a 24 h basis after an appropriate control period for each team. During sleep deprivation subjects were required to perform their assigned military tasks on a sustained basis for the duration of the scenario. Results indicated that their anticipation and perception of the experimental situation affected the common urinary indices of stress. For example, team I, informed that they might be required to sustain operations for 86 h, had significant increases in both urinary 17-hydroxycorticosteroids (17-OHCS) ($p<.01$, 36 h) and total catecholamines ($p<.01$, 24 h). Alternatively, team II, realizing that their maximal deprivation period would not exceed 42 h, had significantly decreased 17-OHCS due largely to decrements in the usually high outputs recorded between 0600-1200 h (e.g. $p<.025$, 30 h). Analogously, total catecholamines were significantly reduced after 24 ($p<.02$) and 30 ($p<.05$) h. We conclude from these studies that under these conditions generally similar effects are noted for sympathicoadrenomedullary and adrenocortical activity. Also, the perception of an anticipated stress (potentially 84 h of sustained operations and sleep deprivation) can neutralize the normally repressive effects of sleep deprivation.

Introduction

For several years we have been interested in the assessment of military task performance and biochemical responses under a variety of stressful environmental conditions including acute exposure to hypobaric hypoxia (5) as well as hot, humid conditions (6). In recent studies we have investigated the effects of sleep deprivation and sustained operations on the responses of two teams of personnel who were performing a simulated military task.

While sleep deprivation has long been known to effect decrements in psychomotor performance (4,7), effects on biochemical indices and physiological responses have been less extensively studied. Tyler et al. (18) and Murawski and Crabbe (10) were unable to demonstrate changes in the excretion of 17-ketosteroids or 17-hydroxycorticosteroids as a result of up to 96 hours of sleep deprivation. Fiorica et al. (4) observed no effects of sleep deprivation on rectal temperature, skin temperature, and oxygen consumption responses upon exposure of the test subjects to acute cold conditions. In fact, these workers concluded generally that sleep deprivation did not elicit the anticipated increments in either adrenocortical or sympathicoadrenomedullary secretion usually identified with physiological stress.

However, in the present study we were interested not only in the effects of sleep deprivation, but also in the additional stress imposed upon the test volunteers by the continuous operations aspect of the study. In addition, our experimental design permitted us to test and compare the responses of two teams, the first of which was informed that the test scenario could persist to a maximum of 86 h, while the second was told that sleep deprivation would not exceed 42 h.

Thus, both teams of trained military personnel were required to perform their assigned military tasks, consisting almost entirely of computation and communication, through open-ended sleep deprivation periods of up to 86 h for team I and 42 h for team II.

Materials and Methods

Test Subjects and Scenario: Two trained artillery fire direction teams, each consisting of five men - an officer, a vertical chart operator, a horizontal chart operator, a radiotelephone operator, and a computer, served as voluntary test subjects. Their tasks consisted largely of receiving coded instruction calls, decoding and plotting of grid coordinates, determining target range and direction, slide-rule computations, and transmitting data to a communications control center. Their performance was evaluated by a team of experts who determined the timeliness and accuracy of their responses. It is important to note that the simulated problems were of consistent difficulty, complexity, and numerical intensity throughout the test scenarios.

During scenario testing the men remained within a stainless steel chamber (22°C, 30-40% RH) and received three hot meals per day. During the establishment of control levels for biochemical constituents and performance, the men remained under restricted dormitory conditions, and slept from 2300-0600 hours daily. From approximately 0730-1600 hours on these control days, Ss remained in the aforementioned chamber performing simulated military problems. During the evening hours of the control days the men were permitted limited activity, consisting of television, games, reading, and a period of exercise. During the sleep deprivation/continuous operations phase of the experiment, physical activity was severely restricted. Team I was informed that they would be expected to sustain operations for a single scenario not to exceed 86 h while team II was told that they might expect two scenarios, neither of which would persist for more than 42 h and the two would be separated by a rest period of at least 33 h.

Sample Collections: During two control days and for the entirety of the experimental scenarios, urine samples were collected. During the sleep deprivation period samples were collected between 0000-0600, 0600-1200, 1200-1800, and 1800-2400. During the control period the evening and overnight samples were slightly altered (1800-2300, 2300-0600) to permit undisturbed sleep. Since intra-individual results did not vary between days for a particular time period, data were combined in a single 24 h control period. Urine samples were assayed for creatinine content according to the method of Bonsnes and Toussky (1), and frozen (-30°C) for subsequent assay. Urine samples were stabilized by the addition of 5 ml of 6N HCl to each collection container.

Assays: Total urinary 17-hydroxycorticosteroids were assayed by a non-hydrolytic method utilizing Steroid-Skreen Kits® (Brinkman Instruments, Inc., Westbury, NY 11590) and measuring hydroxycorticosteroids colorimetrically according to the Porter-Silber method (15). Total urinary catecholamines (free plus conjugated norepinephrine and epinephrine) were assayed utilizing specially prepared Bio-Rad® ion exchange columns as described in their bulletin entitled, "Catecholamines by Column Test". This procedure is based upon the methods of Natelson et al. (11) and Sandler and Freed (14).

Statistical analyses were performed by the t test for paired dependent data, and the null hypothesis was rejected at $p<.05$.

Results

Team I exercised its right to terminate the study after 48 h of sustained operations, and thus the upper half of Figs. 1 and 2 demonstrate results for a 24 h control period followed by a single 48 h sleep deprivation period. The results in the lower sections of Figs. 1 and 2 for team II indicate a 24 h control period followed by two consecutive 36 h sleep deprivation periods; actually, the

two sleep deprivation periods persisted for 39 h and were separated by a 33 h period in which Ss were allowed recovery sleep of up to 12 h during the first night and 8 h during the second night. Since a single test subject in each team failed to maintain adequate urine samples, each figure is based upon the mean ± SEM for four men.

The effects of the combined sleep deprivation/sustained operations on urinary 17-OHCS for teams I and II are depicted respectively in the upper and lower portions of Fig. 1. It can be observed that during the sleep deprivation intervals for both teams the normal periodicity of excretion is disturbed. For team I apparent increases in urinary 17-OHCS midway during the sleep deprivation culminate in a highly significant increase ($p<.01$) at 36 h of sleep deprivation. Alternatively, for team II the lower portion of Fig. 1 indicates generally repressed levels of urinary 17-OHCS during both sleep deprivation intervals with a significant ($p<.025$) decrement noted after 30 h of sleep deprivation during the second scenario. Also, in comparing the urinary excretory rates between the first and second sleep deprivation periods for team II, there again occurred a reduction in 17-OHCS during the latter interval. For example, from 12 to 36 h of the first sleep deprivation scenario, the mean excretion of 17-OHCS is 1.36 mg/6 h/man. During the ensuing sleep deprivation the mean excretion drops to 0.91 mg/6 h ($p<0.01$). During the control interval the average output per man per 6 h period was 1.52 mg.

Fig. 2 depicts generally analogous data for total urinary catecholamines with control levels depicted for each team followed by data from the sleep deprivation/ sustained operations scenarios. In the upper portion of Fig. 2 there is evident a trend toward increasing levels of total catecholamines which is precisely what occurred for 17-OHCS for team I. At 24 h of sleep deprivation the increase is

significant ($p<.01$). The analogy between 17-OHCS and catecholamine excretion for the particular team in question persists for team II as evidenced in the data presented in the lower portion of Fig. 2. For example, after 24 and 30 h of sleep deprivation, there occurred significant decrements in urinary catecholamines during the first scenario ($p<.02$, $p<.05$, respectively); during the second scenario intraindividual variation for one test subject precluded significance.

Discussion

The circadian variation noted in urinary excretion rates of 17-OHCS for both teams is consistent with many that have been published previously (2,3), and is indicative of the early morning burst of adrenocortical activity and release of cortisol which is reflected in a morning and midday peak in metabolite excretion. Generally, the data indicate that for either team the stress of sleep deprivation in combination with sustained operations does not result in marked adrenocortical activation although for team I, a significant increment was noted after 36 h of sleep deprivation; in fact for team II our results seem to be consistent with the hypothesis that subjective fatigue may be associated with a repressive effect on adrenocortical activity. There have appeared in the literature several reports which note a possible repression in adrenocortical activity pursuant to sleep deprivation (10,18); however, the present data were somewhat unexpected due to the level of arousal that was maintained throughout the period of sleep deprivation by the consistent frequency and difficulty of incoming tasks. In an earlier study involving exposure to hypobaric hypoxia with much less rigorous sustained operations, we had also observed indicative, but not significant, trends toward reduced adrenocortical activity (5).

The apparently divergent results noted for the two teams might be explained in terms of an anticipatory or anxiety response among the members of team I who probably felt that they would be unable to complete the rigorous demands of a

prolonged scenario. For example, while team I was mentally prepared for a scenario potentially lasting for 86 h, team II was aware that they would be expected to sustain operations for no more than 42 h. We believe that the differences noted in the excretion of both 17-OHCS and catecholamines are a manifestation in team I of the stress of a novel, and more uncertain situation - continuous operations for 86 h. When the increments in both 17-OHCS and catecholamines were noted, team members, particularly between 24 and 36 h, were probably becoming psychologically stressed by the increasingly apparent feeling that they would be unable to continue for the full 86 h period. Likewise, their level of arousal was probably greatest during the first 36 h, and it has generally been reported that secretion and excretion of catecholamines can be correlated with level of arousal (12,16). Considering the conditions of the test scenarios, the divergent results noted for teams I and II can thus be explained. For example, team I, assuming the need for extended sustained operations/sleep deprivation, demonstrated a higher level of arousal and anxiety through the first 24-36 hours of sleep deprivation. This is manifested in the significant increases noted in urinary 17-hydroxycorticosteroids and catecholamines during this interval. Alternatively, team II, aware of a maximal scenario of 42 h, failed to respond with increased levels of these markers, evidently not perceiving the situation as particularly stressful.

It should be noted that the method employed for urinary catecholamines in the present investigation consistently produces high levels due to the high recovery rates assured by this assay procedure. While our results for excretion of total urinary catecholamines during the control period fell within the ranges reported for normal men, it can be observed that no clear circadian periodic pattern emerged. There are several possible explanations for this which should be considered. Most importantly, Townshend and Smith (17) have reported that the

normal diurnal variation in catecholamine excretion can be repressed by physical activity during the evening hours. In the present experiments the only physical activity allowed Ss during the control period took place in the evening hours probably increasing the normally low excretory rates occurring during sleep. Hartley and his co-workers (9) demonstrated significantly increased levels of plasma norepinephrine at moderate work loads. Similarly, Rodahl and co-workers (13) demonstrated a relationship between daytime physical activity and level of urinary output of catecholamines in fishing industry workmen. The fact that we were measuring total catecholamines may have damped the periodicity since Froberg et al. (7) have demonstrated dissimilar crests of epinephrine and norepinephrine secretion. Winkel and Slob (19) demonstrated reduced periodicity of excretion among younger test subjects as a result of higher nocturnal outputs. Thus, it is clear that the normal excretory pattern of urinary catecholamines is labile and can be altered by a variety of influences.

One related aspect of this study is worthy of further consideration. We have preliminary and limited evidence that following each of the sleep deprivation intervals, several Ss with repressed levels of 17-OHCS and catecholamines responded to a given work load with increased heart rates and $\dot{V}O_2$. Although Fiorica et al. (4) had observed no differences in the physiological responses of sleep deprived men to cold stress, the effects of sleep deprivation on physiological responses to work have not been extensively studied, and it may be worthwhile to investigate further this area with particular reference to the endocrinological responses (8,9) normally resulting from exercise.

Acknowledgement

The views of the authors do not purport to reflect the positions of the Department of the Army or the Department of Defense.

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

The authors are grateful for the expert technical assistance of Mr. Stephen O'Connor and SP Leonard Sakal. We thank Ms Sandra Beach for typing the manuscript. We wish to especially thank the test volunteers for their willingness to participate in these experiments.

Figure Legend

Figure 1 demonstrates the effects of sleep deprivation and sustained operations on the urinary excretion of 17-hydroxycorticosteroids for both FDC teams. Urine was collected during the indicated 6 h intervals. On the left-hand side of both the upper and lower portions are depicted the control levels of 17-OHCS; results for team I (48 h sleep deprivation) are found in the upper half while the lower portion demonstrates results for team II (two 39 h sleep deprivations). Each value represents the mean value ± SEM for 4 men.

Figure 2 illustrates the effects of sleep deprivation and sustained operations in the urinary excretion of total catecholamines in samples collected over 6 h intervals. Again, control data are reported on the left-hand side in either portion; the top half presents data for team I and the bottom portion for team II. Each value represents the mean value ± SEM for 4 men.

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Fig 1

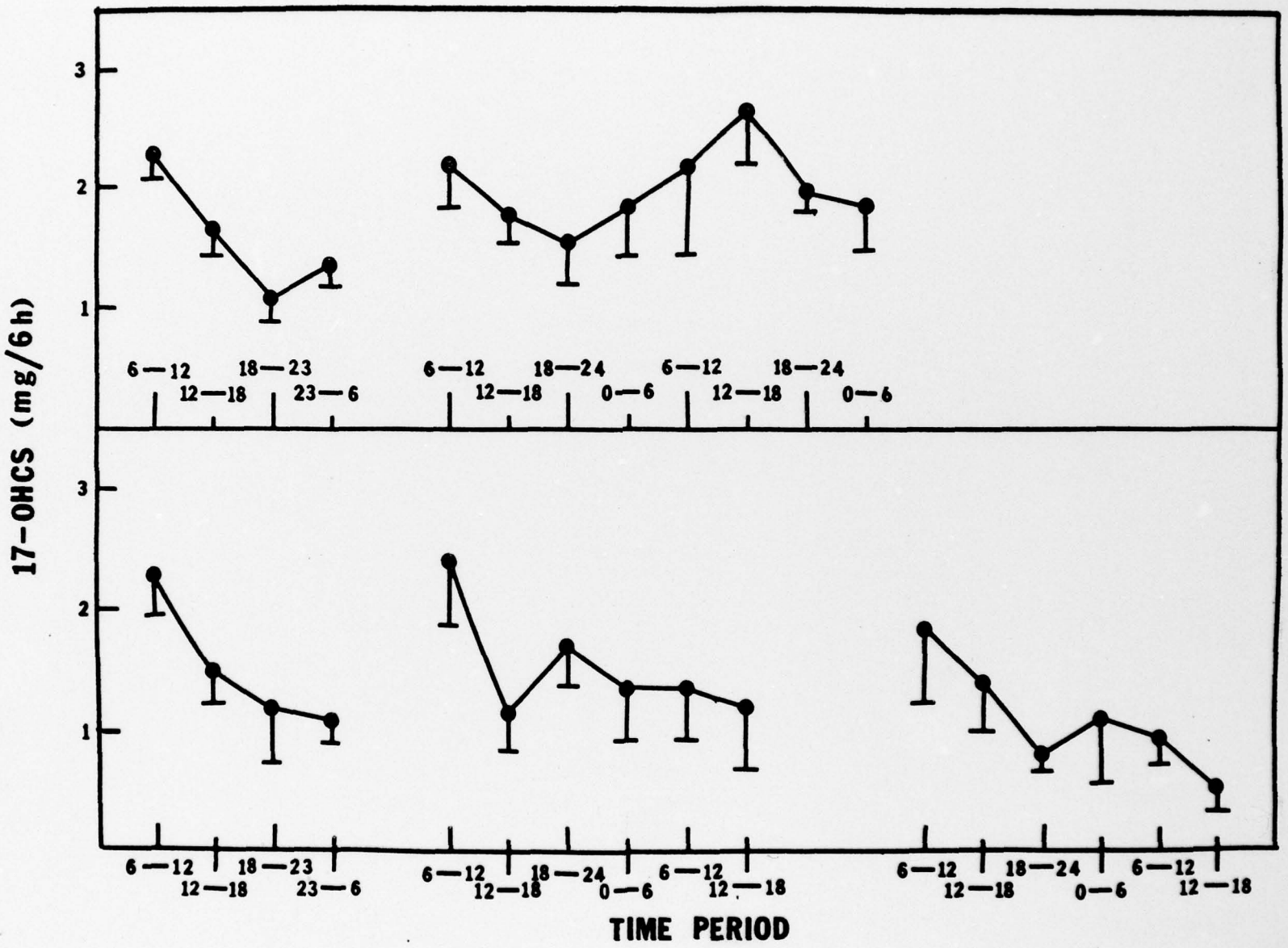
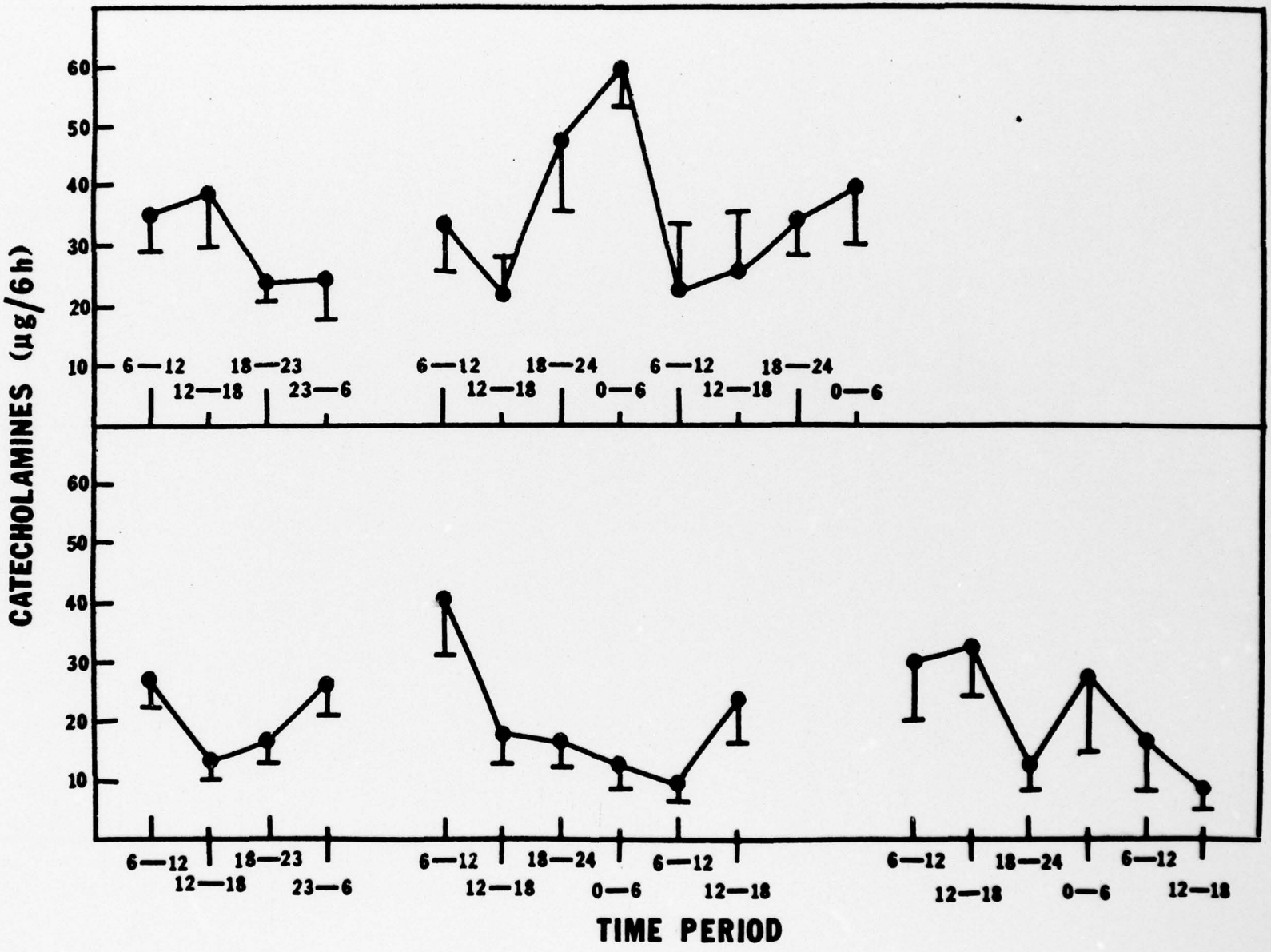


Fig 2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Two groups of highly trained and motivated military personnel were deprived of sleep while sustaining performance of their assigned military tasks in a laboratory simulation; one team (I) was sleep deprived for 48 hours while the second team (II) was deprived of sleep for two consecutive 39 h periods separated by a 33 h rest interval. Six-hour urine samples were collected on a 24 h basis after an appropriate control period for each team. During sleep deprivation subjects were required to perform their assigned military tasks on

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a sustained basis for the duration of the scenario. Results indicated that their anticipation and perception of the experimental situation affected the common urinary indices of stress. For example, team I, informed that they might be required to sustain operations for 86 h, had significant increases in both urinary 17-hydroxycorticosteroids (17-OHCS) ($p<.01$, 42 h) and total catecholamines ($p<.01$, 24 h). Alternatively, team II, realizing that their maximal deprivation period would not exceed 42 h , had significantly decreased 17-OHCS due largely to decrements in the usually high outputs recorded between 0600-1200 h (e.g. $p<.025$, 30 h). Analogously, total catecholamines were significantly reduced after 24 ($p<.02$) and 30 ($p<.05$) h. We conclude from these studies that under these conditions generally similar effects are noted for sympathicoadrenomedullary and adrenocortical activity. Also, the perception of an anticipated stress (potentially 84 h of sustained operations and sleep deprivation) can neutralize the normally repressive effects of sleep deprivation.